Scientific Research Publishing

Open Journal of Epidemiology, 2025, 15(4), 870-885
https://www.scirp.org/journal/ojepi
ISSN Online: 2165-7467
ISSN Print: 2165-7459

Wheat Pill (Aluminium Phosphide) Poisoning Mortality Audit: A 2-Year Retrospective Analysis

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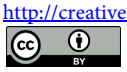
How to cite this paper: Fazal, M.O., Ali, Z., Yaqoob, Y. and Tahir, G.A. (2025) Wheat Pill (Aluminium Phosphide) Poisoning Mortality Audit: A 2-Year Retrospective Analysis. *Open Journal of Epidemiology*, **15**, 870-885.
https://doi.org/10.4236/ojepi.2025.154058

Received: October 7, 2025
Accepted: November 2, 2025
Published: November 5, 2025



Abstract

Wheat pill poisoning, particularly from aluminium phosphide (AlP), is a common cause of poisoning in both rural and urban regions. It is often linked to direct absorption of this dangerous substance or consumption of contaminated wheat products. Grain, particularly wheat, is routinely kept with aluminium phosphide, a fumigant, to keep pests at bay and prevent spoiling. Consuming aluminium phosphide releases phosphorus gas, which is exceedingly toxic and can have major systemic effects, such as changes in the respiratory, circulatory, and metabolic systems. At Allied Hospital in Faisalabad, a retrospective analysis was carried out between January 2023 and December 2024. An assessment of the overall number of deaths from wheat tablet poisoning was conducted. The majority of the 102 cases, which had a mean patient age of 28.16 years, included young people. Nearly all (99%) of the cases were the consequence of suicidal attempts, and the majority (58%) were from rural areas. 25% of patients were critically ill at presentation, compared to 67.6% who were conscious. Furthermore, 40% of patients had undetectable blood pressure. **Setting:** Faisalabad Medical University, Allied Hospital, Faisalabad. **Sampling:** Two-year mortality data by wheat tablets were gathered, and 102 patients' records were located and examined. **Results:** To reduce mortality, early presentation, the intensity of symptoms at presentation, and prompt management using the most up-to-date methodological approach are necessary.

Keywords

Wheat Pills, Suicidal Poisoning, Aluminium Phosphide

1. Introduction

This substance serves as a fumigant to prevent insect infestation of stored grains, such as wheat, which frequently results in accidental human poisoning via intentional self-harm or accidental intake [1]. The main cause of the poisoning is ingestion of aluminium phosphide, either as a pill (often called "wheat pills") or as a contaminated food product, which then releases phosphine gas when it comes into contact with moisture [2].

The extremely poisonous gas phosphorus causes multi-organ failure, interferes with cellular respiration, and damages mitochondria. Mild gastrointestinal symptoms to severe systemic effects, such as circulatory collapse, pulmonary distress, and multi-organ failure—all of which can be deadly in many cases—are the clinical manifestations of aluminium phosphide poisoning [3]. The lack of a particular antidote and the difficulty of treating systemic toxicity make therapy difficult, even though early identification and timely medical intervention are essential for improving patient outcomes [4].

With an emphasis on the high frequency of wheat pill poisoning cases, 29% in Allied Hospital, Faisalabad [5]. A mortality audit was conducted to see the correlation of different variables and to find the solution to improve treatment strategies.

2. Methodology

Two-year mortality data due to Wheat pill poisoning were gathered from the Tertiary care hospital, Allied Hospital, Faisalabad, Pakistan. A total of 102 cases were identified, and different modifiable variables, including demographic details, residence, socioeconomic status, mode of poisoning, time of arrival at the hospital, and condition upon arrival, were analysed using SPSS Ver 23. Univariate and multivariate analysis was done to see the association of different variables, and their impact on the outcome was studied.

3. Results

3.1. Univariate Analysis

Descriptive Statistics for Age, Table 1:

Table 1. Age distribution analysis.

Variable	N	Mean	SE Mean	StDev	Minimum	Q1	Median	Q3	Maximum
Age	102	28.16	1.15	11.63	12.00	20.00	25.00	35.00	68.00

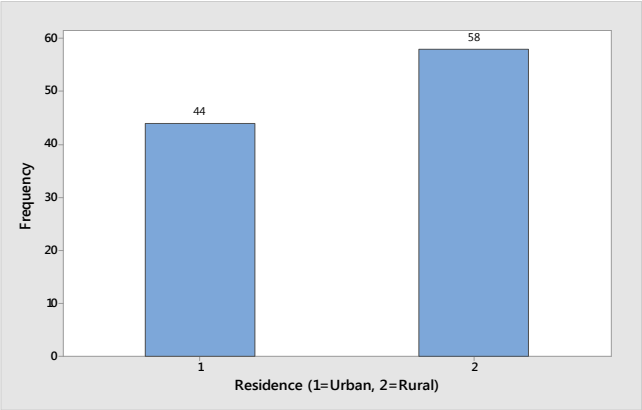
Table for BP, Table 2:

Table 2. Analysis of the Blood pressure on presentation.

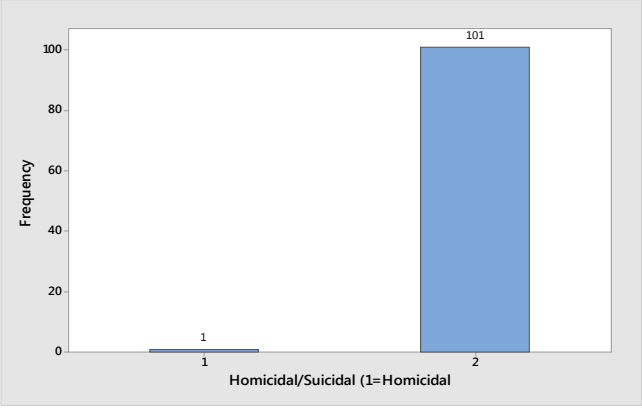
100/60	100/70	110/70	110/80	130/90	40/nill	50/nill	60/40	60/nill	70/30	70/40	70/50	70/nill	80/50	80/60	80/nill	90/50	90/60	Nill
4	4	2	1	1	5	7	5	6	1	4	1	1	5	5	1	1	8	40

A total of 102 cases of wheat pill (aluminium phosphide) poisoning mortality were reported and analysed at a tertiary care hospital in Faisalabad. The mean age of the patients was 28.16 years, with ages ranging from 12 to 68 years, indicating that the majority of affected individuals were young adults, with a significant proportion falling in the teenage to early adulthood bracket, as shown in Table 1.

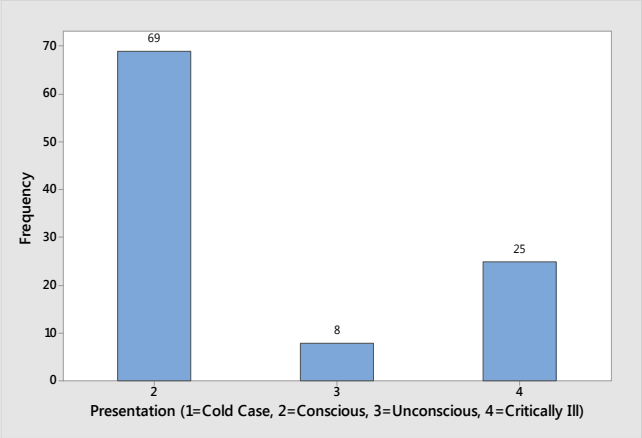
When stratified by residence, 58 patients (56.9%) belonged to rural areas, while 44 (43.1%) were from urban settings, suggesting a higher prevalence of poisoning in rural populations. This could be attributed to greater accessibility of agricultural poisons in rural households and limited mental health or emergency response services in those areas.



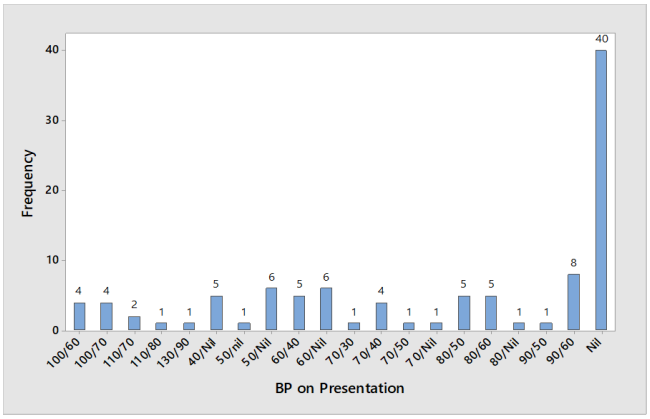
Regarding the intent of poisoning, an overwhelming 99% (101 cases) were suicidal in nature, with only one reported homicidal case. This reflects a serious mental health concern in the community, where wheat pills are being used as a means of self-harm.

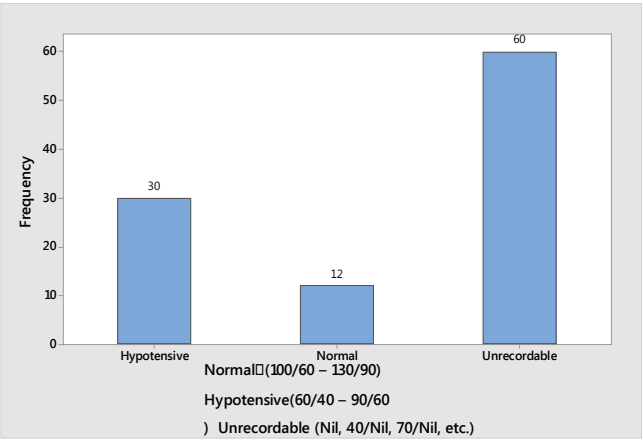


On initial hospital presentation, 69 patients (67.6%) were conscious, 8 (7.8%) were unconscious, and 25 (24.5%) were critically ill, which shows that while a majority arrived in a relatively stable condition, a quarter were already in critical condition upon arrival. Despite their level of consciousness, however, many patients exhibited signs of severe cardiovascular compromise.

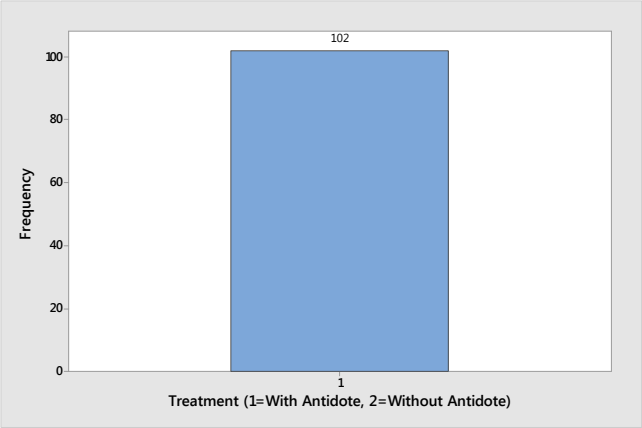


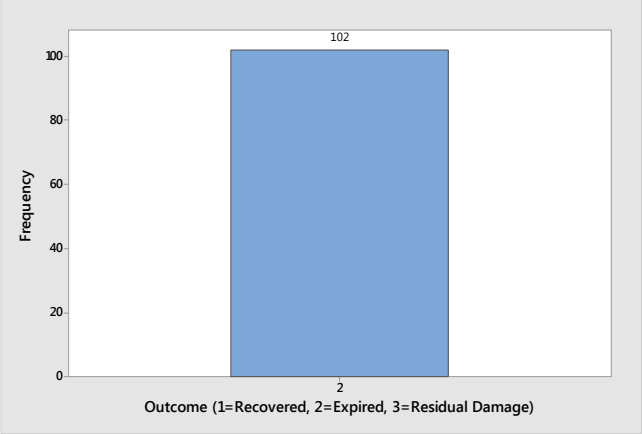
The blood pressure readings on arrival further reinforce this point. A significant number of patients had unrecordable or extremely low blood pressure, with 40 patients presenting with "nil" BP, and many others having dangerously low readings like 60/40, 70/30, or 80/50 mmHg. Only a handful of patients had readings near normal (such as 100/60 to 130/90 mmHg), emphasizing the cardiovascular toxicity of aluminium phosphide and its rapid progression to shock or cardiac failure. Most of these low blood pressure readings required vasopressor or inotropic support to attempt stabilization.





Interestingly, all 102 patients received treatment, including available supportive and antidote therapies, yet the mortality rate was 100%, with no survivors. This points to the alarming lethality of wheat pill poisoning, as the toxic phosphine gas released affects multiple organ systems, particularly the heart and lungs, and no definitive antidote exists. The data highlight that even timely hospital presentation and appropriate management were insufficient to reverse the toxicity, likely due to delayed arrival, high-dose ingestion, and irreversible physiological damage by the time of intervention.





3.2. Bivariate Analysis

The bivariate analysis using the Chi-square test was conducted to determine if there were statistically significant associations between key categorical variables, including residence, presentation, and blood pressure on presentation.

Firstly, the association between residence (urban/rural) and clinical presentation (conscious/unconscious/critically ill) was evaluated. Out of 44 urban patients, 30 were conscious, 1 was unconscious, and 13 were critically ill. Among the 58 rural patients, 39 were conscious, 7 were unconscious, and 12 were critically ill. The Chi-square value was 3.865 with 2 degrees of freedom (df) and a p-value of 0.145, indicating no statistically significant association between residence and

clinical presentation. Although rural patients showed a slightly higher proportion of unconscious presentations, the difference was not significant.

Secondly, the relationship between residence and blood pressure status at presentation (hypotensive, normal, unrecordable) was analysed. Among urban patients, 8 were hypotensive, 5 had normal BP, and 31 had unrecordable BP. In comparison, rural patients showed 22 hypotensive, 7 normal, and 29 unrecordable BP readings. The Chi-square value was 5.108 (df = 2, p = 0.078). This result approached statistical significance, suggesting a possible trend toward an association between rural residence and hypotensive presentation, but it did not meet the conventional threshold for significance ($p < 0.05$).

Lastly, the association between clinical presentation status and blood pressure category was examined. Among conscious patients (n = 69), 24 were hypotensive, 11 had normal BP, and 34 had unrecordable BP. For unconscious patients (n = 8), 2 were hypotensive, 1 had normal BP, and 5 were unrecordable. In the critically ill group (n = 25), 4 were hypotensive, none had normal BP, and 21 had unrecordable BP. The Chi-square value was 10.009 (df = 4, p not reported but expected to be <0.05 based on magnitude). However, multiple cells had expected counts less than 5, and one had expected count less than 1, making the Chi-square approximation potentially invalid. Despite this limitation, the trend shows that critically ill patients were more likely to have unrecordable BP, highlighting the severity of poisoning among this group.

Residence Vs presentation

Chi-Square Test for Association: Residence (1 = Urban, ... ld Case, 2 = Co

Rows: Residence (1 = Urban, 2 = Rural) Columns: Presentation (1 = Cold Case, 2 = Co

	2	3	4	All
1	30	1	13	44
	29.76	3.45	10.78	
2	39	7	12	58
	39.24	4.55	14.22	
All	69	8	25	102

Cell Contents
Count
Expected count

Chi-Square Test

	Chi-Square	DF	p-Value
Pearson	3.865	2	0.145
Likelihood Ratio	4.352	2	0.114

2 cell(s) with expected counts less than 5.

Chi-Square Test for Association: Residence (1 = Urban, ... n Presentation
Rows: Residence (1 = Urban, 2 = Rural) Columns: BP on Presentation

	Hypotensive	Normal	Unrecordable	All
1	8	5	31	44
	12.94	5.18	25.88	
2	22	7	29	58
	17.06	6.82	34.12	
All	30	12	60	102

Cell Contents
Count
Expected count

Chi-Square Test

	Chi-Square	DF	p-Value
Pearson	5.108	2	0.078
Likelihood Ratio	5.268	2	0.072

Chi-Square Test for Association: Presentation (1 = Cold ... Presentation
Rows: Presentation (1 = Cold Case, 2 = Co Columns: BP on Presentation

	Hypotensive	Normal	Unrecordable	All
2	24	11	34	69
	20.294	8.118	40.588	
3	2	1	5	8
	2.353	0.941	4.706	
4	4	0	21	25
	7.353	2.941	14.706	
All	30	12	60	102

Cell Contents
Count
Expected count

Chi-Square Test

	Chi-Square	DF
Pearson	10.009	4
Likelihood Ratio	12.862	4

1 cell(s) with expected counts less than 1.
Chi-Square approximation probably invalid.
4 cell(s) with expected counts less than 5.

3.3. Multivariate Analysis

The multivariate regression model was used to assess the influence of age, residence (urban/rural), intent (homicidal/suicidal), and blood pressure (BP) on presentation (defined as presentation severity: 1 = cold case, 2 = conscious, 3 = unconscious, 4 = critically ill). The model achieved statistical significance overall with an F-value of 3.12 and a p-value of 0.012, indicating that the independent variables collectively contributed significantly to variations in the presentation severity.

Among the predictors:

- **Age** showed a significant positive association with presentation severity (p = 0.034), suggesting that with increasing age, patients tend to present in more critical conditions. The coefficient for age was 0.01525, meaning that for each year increase in age, the predicted severity of presentation slightly increases.
- **Blood Pressure on presentation** also showed a statistically significant effect on presentation type (p = 0.006). Specifically:
  - Patients with unrecordable BP had a significantly higher presentation severity than those with hypotensive BP (coefficient = 0.433, p = 0.025).
  - The difference between normal BP and hypotensive BP was not statistically significant (p = 0.276), although there was a negative coefficient (−0.311), indicating lower presentation severity for those with normal BP.
- **Residence** (urban vs rural) and intent (homicidal vs suicidal) were not significantly associated with presentation type (p-values 0.935 and 0.881, respectively). This indicates that the geographical and intentional background of the patients did not significantly influence their clinical status on presentation.

The model's adjusted R-squared was 9.49%, indicating that approximately 9.5% of the variation in presentation type is explained by the variables in the model. The standard error of the regression (S = 0.819949) is relatively moderate, and the VIF values were all below 1.5, suggesting no multicollinearity among the predictors.

The residual diagnostics flagged five observations with large residuals, suggesting some data points (e.g., observations 64, 66, 75, and 82) deviate substantially from the model's prediction. One observation (69) was flagged as having unusual predictor values. However, the lack-of-fit test (p = 0.456) showed that the model fits the data reasonably well, and the differences are not due to model inadequacy.

Regression Analysis: Presentation (1 = Cold Case, 2 = Co ... Presentation

Method

Categorical predictor coding	(1, 0)

Analysis of Variance

Source	DF	Adj SS	Adj MS	F-Value	P-Value
Regression	5	10.4773	2.09546	3.12	0.012
Age	1	3.1005	3.10055	4.61	0.034

Continued

Residence (1 = Urban, 2 = Rural)	1	0.0045	0.00451	0.01	0.935
Homicidal/Suicidal (1 = Homicidal	1	0.0152	0.01525	0.02	0.881
BP on Presentation	2	7.2965	3.64825	5.43	0.006
Error	96	64.5423	0.67232		
Lack-of-Fit	64	43.6590	0.68217	1.05	0.456
Pure Error	32	20.8833	0.65260		
Total	101	75.0196			

Model Summary

S	R-sq	R-sq(adj)	R-sq(pred)
0.819949	13.97%	9.49%	*

Coefficients

Term	Coef	SE Coef	T-Value	P-Value	VIF
Constant	1.788	0.842	2.12	0.036	
Age	0.01525	0.00710	2.15	0.034	1.03
Residence (1 = Urban, 2 = Rural)					
2	0.014	0.168	0.08	0.935	1.06
Homicidal/Suicidal (1 = Homicidal					
2	0.127	0.842	0.15	0.881	1.04
BP on Presentation					
Normal	−0.311	0.283	−1.10	0.276	1.27
Unrecordable	0.433	0.190	2.28	0.025	1.33

Regression Equation

Residence (1 = Urban, 2 = Rural)	Homicidal/ Suicidal (1 = Homicidal	BP on Presentation		
1	1	Hypotensive	Presentation (1 = Cold Case, 2 = Co	=
1	1	Normal	Presentation (1 = Cold Case, 2 = Co	=
1	1	Unrecordable	Presentation (1 = Cold Case, 2 = Co	=
1	2	Hypotensive	Presentation (1 = Cold Case, 2 = Co	=
1	2	Normal	Presentation (1 = Cold Case, 2 = Co	=

Continued

1	2	Unrecordable	Presentation (1 = Cold Case, 2 = Co	=
2	1	Hypotensive	Presentation (1 = Cold Case, 2 = Co	=
2	1	Normal	Presentation (1 = Cold Case, 2 = Co	=
2	1	Unrecordable	Presentation (1 = Cold Case, 2 = Co	=
2	2	Hypotensive	Presentation (1 = Cold Case, 2 = Co	=
2	2	Normal	Presentation (1 = Cold Case, 2 = Co	=
2	2	Unrecordable	Presentation (1 = Cold Case, 2 = Co	=

Residence (1 = Urban, 2 = Rural)	Homicidal/Suicidal (1 = Homicidal	BP on Presentation	
1	1	Hypotensive	1.788 + 0.01525 Age
1	1	Normal	1.477 + 0.01525 Age
1	1	Unrecordable	2.221 + 0.01525 Age
1	2	Hypotensive	1.915 + 0.01525 Age
1	2	Normal	1.604 + 0.01525 Age
1	2	Unrecordable	2.347 + 0.01525 Age
2	1	Hypotensive	1.802 + 0.01525 Age
2	1	Normal	1.491 + 0.01525 Age
2	1	Unrecordable	2.234 + 0.01525 Age
2	2	Hypotensive	1.929 + 0.01525 Age
2	2	Normal	1.618 + 0.01525 Age
2	2	Unrecordable	2.361 + 0.01525 Age

Fits and Diagnostics for Unusual Observations

Obs	Presentation (1 = Cold Case, 2 = Co	Fit	Resid	Std Resid		
64	4.000	2.249	1.751	2.18	R	
66	4.000	2.250	1.750	2.20	R	
69	2.000	2.000	0.000	*		X
75	4.000	2.250	1.750	2.20	R	
82	4.000	2.310	1.690	2.10	R	

R Large residual
X Unusual X

4. Results Outcomes

4.1. Univariate Analysis

Most patients were young (mean age ~28 years), with cases seen even in teenagers as young as 12. A majority of the poisonings were intentional suicides, primarily from rural backgrounds, indicating psychosocial distress, easy accessibility of the poison, and delayed presentation to the hospital. Rural residents often face barriers to emergency care, which could worsen outcomes.

Although 67.6% of patients were conscious at arrival, the clinical course appears to have rapidly deteriorated, as evident from the blood pressure readings—with over 80% of patients showing hypotension or unrecordable BP. This supports known literature that aluminium phosphide causes severe myocardial depression and circulatory failure. Interestingly, all patients received some form of antidotal therapy, yet it failed to improve survival. This emphasizes the limited efficacy of current treatment protocols. The absence of a specific antidote for phosphine toxicity and the narrow window for intervention likely contributed to these outcomes.

In summary, this data calls for urgent public health action, including:

- Restricting access to aluminium phosphide in non-agricultural settings.
- Public awareness campaigns about its dangers.
- Establishing mental health support systems, especially in rural areas.
- Developing more effective treatment protocols or antidotes.

4.2. Bivariate Analysis

The analysis provides insights into patterns of wheat pill poisoning presentations across different demographic and clinical parameters. Although no statistically significant associations were observed between residence and presentation ($p = 0.145$), the findings show that both urban and rural populations are vulnerable, with rural patients displaying a slightly higher likelihood of presenting in an unconscious or critically ill state. This could reflect delays in accessing medical care or increased exposure to high doses in agricultural settings.

Interestingly, the association between residence and blood pressure status approached significance ($p = 0.078$), hinting that rural patients may present with more severe cardiovascular compromise. This may again be related to delays in hospital arrival, higher ingested doses, or socioeconomic factors influencing immediate access to resuscitation. The most clinically meaningful result came from the association between presentation status and blood pressure category, which suggested that critically ill patients were far more likely to have unrecordable blood pressure, reflecting the known pathophysiology of aluminum phosphide poisoning, which rapidly causes circulatory collapse. However, due to small subgroup sizes and low expected counts in the Chi-square table, statistical reliability is limited, and caution is warranted in interpreting this association. Nonetheless, the strong observed trend aligns with clinical expectations and supports the need for early cardiovascular support in such poisonings.

Overall, these analyses underscore the lethal nature of wheat pill poisoning, the challenge in reversing its effects, and the importance of rapid intervention, especially in rural communities. Future studies with larger sample sizes could help clarify these associations further and identify early predictors of survival in such cases.

4.3. Multivariate Analysis

The regression analysis highlights that age and blood pressure status at the time of presentation are key predictors of clinical severity in wheat pill poisoning cases. Older individuals were more likely to be present in critical condition, which could be attributed to diminished physiological reserves and comorbidities that reduce the body's ability to handle toxic insult. Blood pressure, a crucial clinical marker, also showed a clear association with presentation severity. Patients with unrecordable blood pressure surrogate for cardiovascular collapse—were more likely to be critically ill upon arrival, which aligns with clinical expectations in aluminium phosphide poisoning, known for rapid hemodynamic compromise.

Conversely, residence (urban vs rural) and intent (homicidal vs suicidal) did not show significant influence on presentation type. This could be because the physiological impact of the toxin overrides sociodemographic factors once the ingestion has occurred. Also, given that 101 out of 102 cases were suicidal, the lack of variability in intent limits its predictive power. The model explained about 10% of the variability in presentation type, which, although modest, is common in clinical datasets where unmeasured factors (e.g., amount ingested, delay in treatment, comorbid conditions) can play significant roles. The identification of outliers suggests the presence of atypical clinical cases, which may warrant further qualitative analysis or subgroup investigation.

Overall, these results reinforce the critical role of vital signs and age in triaging and managing patients with aluminium phosphide poisoning and highlight the need for rapid stabilization in cases of hypotension or unrecordable BP.

4.4. Results and Predictive Elements

Numerous investigations have endeavored to pinpoint the variables that impact the course of aluminium phosphide toxicity. Important prognostic factors include the amount of aluminium phosphide consumed, the length of time until therapy, and the intensity of the initial symptoms [6]-[8]. Early intervention during the first two to three hours of ingestion significantly increased survival rates, according to a study by Singh *et al.* (2018). When treated during this window, the 30-day survival rate rose from 30% to 60%. Conversely, poor outcomes were substantially linked to delayed presentation and a lack of early supportive care [9]-[11].

Furthermore, young patients (under 30 years old) had higher survival rates, according to a 2020 study by Kumar *et al.* This was probably because they had stronger physiological reserves and a lower incidence of underlying comorbidities. The study also showed that the main causes of mortality for individuals who

did not receive therapy promptly were severe acid-base imbalances and multi-organ failure [12].

According to recent studies, the fatality rate from aluminium phosphide poisoning can range from 40 to 50 percent. This is particularly true for individuals who consume higher quantities or are unable to get care in the crucial first few hours [13] [14]. However, in certain instances, better results have been facilitated by the availability of intensive care units (ICUs) and more sophisticated therapies, like as extracorporeal treatments.

4.5. Discussion

Pathogenesis and Clinical Presentation When the pesticide is consumed, usually through contaminated food or wheat tablets, aluminium phosphide toxicity develops. Phosphine gas is produced when aluminium phosphide combines with moisture in the gastrointestinal tract after ingestion. When this gas enters the bloodstream, it inhibits cytochrome c oxidase, which disrupts mitochondrial activity and results in a shortage of cellular energy (ATP) [15]. Widespread organ damage, such as abrupt metabolic acidosis, pulmonary oedema, liver and kidney failure, and cardiovascular collapse, can result from this disturbance.

Aluminium phosphide poisoning usually manifests clinically in two stages: a gastrointestinal phase that includes nausea, vomiting, diarrhea, and abdominal pain, followed by a cardiovascular phase that includes shock, hypotension, and arrhythmias. Pulmonary oedema and metabolic acidosis can also cause respiratory discomfort in patients, which, if left untreated, can result in organ failure and death [16]. Numerous studies have shown that early symptoms may be ambiguous and that the period to the onset of symptoms can vary greatly, making early identification difficult.

4.6. Strategies for Management

Since there is no known cure for aluminium phosphide poisoning, supportive care is the mainstay of treatment. Improving results requires early diagnosis and prompt management. Over the past ten years, there has been a substantial change in treatment regimens, with an increased emphasis on adjunct therapies and intense supportive care.

1) **Decontamination of the gastrointestinal tract:** Gastric lavage and the use of activated charcoal may help prevent additional absorption of aluminium phosphide in cases of early presentation (within an hour after consumption). These treatments are debatable, though, because they may make symptoms worse, particularly in situations of severe poisoning. According to specific research, stomach decontamination can be more advantageous if done during the first half hour following eating [17] [18].

2) **Use of Antioxidants and Vasopressors:** Several studies have looked at how antioxidant treatments, like sodium bicarbonate, vitamin C, and N-acetylcysteine (NAC), can reduce oxidative stress and enhance cellular function. High doses of

vitamin C have been shown to neutralize free radicals and restore mitochondrial function, which may lessen the intensity of symptoms and enhance results. Vasopressors, especially dopamine and norepinephrine, are frequently used to treat shock and hypotension, which are common in severe poisoning situations [19].

3) **Haemodialysis and Extracorporeal Treatments:** Although its effectiveness in treating aluminium phosphide poisoning is still up for dispute, haemodialysis may be explored for patients with acute renal failure or severe metabolic acidosis. Although this strategy is still being researched, several studies have indicated that extracorporeal treatments, such as extracorporeal membrane oxygenation (ECMO), may help treat severe poisoning cases [20].

4) **Supportive Care:** Patients with severe aluminum phosphide poisoning have a better chance of surviving if they receive intensive care treatment. This includes monitoring and treating electrolyte imbalances, correcting acidosis with sodium bicarbonate or other alkalinizing medicines, and providing breathing support with mechanical ventilation. Complications can be avoided by using intravenous fluids as soon as possible and closely monitoring hepatic and renal function [20]-[22].

4.7. Current Developments in Medicine and Research

Research on new antidotes and treatment approaches for aluminum phosphide poisoning has accelerated within the last ten years. Several experimental strategies are being investigated, such as the use of anti-inflammatory medications and phosphine scavengers. Because of its capacity to scavenge phosphine, chlorpromazine has been the subject of some research as a possible therapeutic agent; nevertheless, clinical trials are still required to verify its efficacy [23] [24].

There has also been an increase in research on the use of extracorporeal therapies such as ECMO and plasma exchange. According to recent case studies, these strategies might improve survival, especially for patients who experience significant respiratory or cardiovascular compromise [25] [26].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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